

The Anti-Aging Elixir?

Researchers have identified CLCF1, a crucial protein that declines with age but is released during physical activity, playing a central role in protecting muscles and bones from age-related deterioration. This discovery could unlock new strategies to reverse the effects of aging.

By National Research Council of Science & Technology

It's a widely accepted truth that "[exercise is good for your health](#)," but the precise molecular mechanisms behind this phenomenon have often remained a mystery. Now, a collaborative research effort, spearheaded by Dr. Yong Ryoul Yang of [the Aging Convergence Research](#) Group at the Korea Research Institute of Bioscience and Biotechnology (KRIBB) and Professor Nak-Sung Kim of Chonnam National University, has pinpointed a significant piece of this puzzle: a protein named **CLCF1 (cardiotrophin-like cytokine factor 1)**.

This newly characterized myokine (a protein released by muscle cells) appears to be a central conductor of exercise's myriad health benefits. The team's findings reveal that CLCF1 is released by muscles during physical activity, where it actively supports both [muscle and bone strength](#), ultimately contributing to the deceleration of musculoskeletal aging.

CLCF1: A Key Player in Aging and Exercise Response

To understand how CLCF1 responds to the twin forces of aging and exercise, the researchers conducted a comparative study. They divided participants into young and elderly groups and measured their blood levels of CLCF1 after physical activity. The results were telling: younger individuals exhibited a significant surge in CLCF1 levels after just a single [exercise session](#). In stark contrast, older adults required a more prolonged commitment, showing increased CLCF1 levels only after more than 12 weeks of consistent exercise.

Further compelling evidence emerged from experiments with aged mice. When CLCF1 was directly administered to these mice, they displayed remarkable improvements in muscle strength and a notable increase in bone density. Conversely, when CLCF1 activity was deliberately blocked, the positive effects of exercise were entirely negated. This crucial finding unequivocally confirms CLCF1's essential role in the body's adaptive response to [physical exertion](#).

Unlocking Molecular Mechanisms and Therapeutic Potential

Delving deeper into the molecular mechanisms, the researchers discovered that CLCF1 works on multiple fronts. It actively enhances mitochondrial function within [muscle cells](#), mitochondria being the "powerhouses" of the cells. Furthermore, CLCF1 inhibits the formation of osteoclasts (cells that resorb bone) while simultaneously promoting the differentiation of osteoblasts (cells responsible for bone formation).

This groundbreaking research provides the first scientific evidence identifying changes in protein secretion, specifically the decline of CLCF1, as a major underlying reason for why the efficacy of exercise tends to diminish in aging individuals.

"This research provides [a biological basis](#) for why exercise becomes less effective with age, and it lays the groundwork for developing new therapeutic strategies for healthy aging," stated Dr. Yong Ryoul Yang from KRIBB. He added, "In particular, the findings offer new directions for treating age-related sarcopenia [muscle loss] and osteoporosis [bone loss]."

This discovery of CLCF1 not only deepens our understanding of the profound [benefits of exercise](#) but also opens exciting new avenues for developing interventions that could potentially reverse age-related decline in musculoskeletal health, offering a promise of healthier, more active aging.